Attachment 2

THE AGENCY'S RESPONSE TO COMMENTS ON THE TERRESTRIAL RISK ASSESSMENT FOR METHYL PARATHION

Summary: Cheminova disputes the results of EPA's risk assessment primarily by contesting the validity of EPA's risk index method. While Cheminova commented on several aspects of the risk assessment particular to methyl parathion, their comments center mainly on the following aspects of EPA's standard methods:

- The use of the lowest toxicity endpoint for the calculation of risk quotients (RQs) for a particular class of non-target animals;

- The use maximum rates, and number and frequency of applications, in calculating expected environmental concentrations (EECs);

- The choice of feed items for birds and mammals, and the use of Kenaga (1973) and Fletcher (1994) nomographs for estimating exposure through consumption of contaminated food items; and

- The comparison of maximum, day-0 EECs to acute and chronic toxicity endpoints for assessment of risk.

Cheminova provided an alternative risk assessment in their comments which incorporated what they believe to be more "appropriate" toxicity endpoints and estimated exposure values. While their calculations resulted in lower RQ values, their results still indicated level-of-concern (LOC) exceedences for several classes of birds and mammals. The registrant also compared their toxicity endpoints to methyl parathion residues measured in field trials. This resulted in even fewer LOC exceedences although some remained.

The World Wildlife Fund, with the support of other non-governmental organizations such as the Consumers Union and Natural Resources Defense Fund, provided a set of open literature articles as evidence that methyl parathion may cause endocrine disruption in non-target organisms. EPA recommends that methyl parathion be subject to definitive testing to further investigate the potential for these effects.

In response to comments on the preliminary risk assessment, EPA has made several revisions to the terrestrial risk assessment for methyl parathion. For instance, discussion of potential bioaccumulation of methyl parathion in avian food items has been removed because the likelihood of effects to birds via this pathway was not compelling. In addition, language suggesting that risk to beneficial insects in general is equivalent to that posed to honeybees has been removed, because EPA does not have evidence to support such a conclusion.

The conclusions of the terrestrial risk assessment remain essentially the same as they were described in the preliminary risk assessment. One exception is the **expected reduced risk to bees associated with the voluntary cancellation of tree fruit uses of methyl parathion.** Most changes made in response to Cheminova's comments were clarifications to the risk assessment, or corrections of internal inconsistencies in the document. The original risk assessment was based on scientifically-sound data submitted to the Agency to meet registration requirements for methyl parathion, and was well-supported by incident data and the open literature.

Specific comments received on the terrestrial risk assessment include:

Cheminova Comment: EPA should admit that LOC's in the risk index method of risk assessment represent only a screen. "Exceedences should only be interpreted as meaning that more in-depth assessments need to be undertaken."

EPA Response: There could be refinements based on technically valid and statistically robust studies that would address exposure and effects uncertainties. However, these refinements would require that a number of uncertainties both for exposure and effects be quantified. This would require that additional data be submitted. The Agency is currently exploring ways to improve ecological assessments (ECOFRAM), to provide a mechanism through which these uncertainties can better be addressed. At the present time, EPA believes that the available incident data and effects data from the open literature support the conclusions presented in the preliminary risk assessment.

Cheminova Comment: Cheminova believes it is inappropriate to search the "available database for the lowest available (toxicity endpoint) value and then use that value to calculate point estimate risk quotients." They used "calculated mean values and 95% confidence limits using standard statistical procedures and the value of the lower 95% confidence limit was selected as the dietary toxicity endpoint." Cheminova separated quail and mallard data in their assessment because the toxicity values for these are different by an order of magnitude. They compared bobwhite to grass and leaf EECs, and mallard to insects and seeds/fruits.

EPA Response: Only two bird species are tested, one waterfowl species and one upland gamebird species, under the Fish and Wildlife Data Requirements listed in CFR 158. There is a great deal of uncertainty associated with extrapolating from the acute oral and subacute dietary data from two species to the large numbers of bird species associated with agricultural areas. Our ecological risk assessments assume that if we use the most sensitive tested species in the laboratory, then we provide some protection to untested species in the environment.

Field surveys indicate that a large variety of birds are associated with agricultural systems. The EPA ecological database indicates that there can be a large variation in sensitivity to a chemical between species. Using the most sensitive species tested and upper limits of estimated environmental concentrations to calculate risk is a reasonable way to compensate for this uncertainty in the toxicity data.

The basis for Cheminova's approach to data evaluation for selecting toxicity endpoints is not clear. They indicate that the entire data set should be considered and that this is done by calculating the mean values and the 95% confidence limits using standard statistical procedures. The value of the lower 95% confidence limit was then selected as the dietary toxicity endpoint. Cheminova's approach appears to assume that toxicity estimates are not species specific, allowing the pooling of data. However, they separate the mallard from other species due to its very distinct difference in sensitivity. This would suggest that the pooling o toxicity data would be inappropriate for the other species as well, but no explanation is given beyond the difference in sensitivity. This difference in sensitivity, however, is one of the main reasons that pooling of toxicity data is inappropriate and can lead to erroneous conclusions in the assessment of risk.

Cheminova Comment: "Cheminova does not believe that standard, longer-term (chronic) risk, as evaluated using EPA's longer-term guideline studies, is applicable to methyl parathion because of its use pattern and very short environmental persistence. Rather, any risks associated with methyl parathion use are more appropriately evaluated as acute or subacute risks.

"Even under the maximum application scenarios, which involve up to 6 to 10 applications to a crop, the duration of the exposure scenario is only about 4 to 6 weeks because the applications occur over very short intervals under these maximum application scenarios. Thus the "longer-term" exposures are most appropriately considered subacute to subchronic exposures."

EPA Response: Existing toxicological studies are limited in their ability to distinguish between latent expression of adverse effects from short exposures and those effects requiring more protracted exposure for expression. In other words, the available toxicity studies can not determine how long organisms must be exposed before effects occur. In addition, data from the open literature suggest that methyl parathion may cause long-term effects through endocrine disruption, further exemplifying the limits in the ability of existing toxicity studies to distinguish latent expression of adverse effects from short exposures.

Cheminova Comment: EPA's avian and mammalian risk assessments assume that animals will eat only one kind of food, and that all of the food consumed will be contaminated with methyl parathion at the maximum initial estimated residues.

EPA Response: The current risk assessment method for terrestrial organisms was designed to account for uncertainties. Because of the uncertainty, the method includes some conservative assumptions. In some aspects, such as those described above, conservative assumptions are made that will tend to over-estimate exposure. However, in many other aspects, assumptions are made and factors are overlooked that may cause the risk to be underestimated. For example, all routes of exposure other than ingestion of contaminated food are ignored, and animals in the wild are assumed to consume food at a rate no greater than those kept in captivity. Overall, the Agency does not believe that the risk assessment is inappropriately conservative. The Agency is working on developing risk assessment methods that will address some of these factors. The Agency is open to receiving data from Cheminova which further explores consumption patterns of terrestrial organisms in the wild.

Cheminova Comment: Birds and mammals found in treated field will most likely feed on insects, seeds and soil invertebrates. The "risk analysis should be weighted to reflect the higher probability of these types of feed items being ingested by birds and small mammals in agricultural settings."

EPA Response: Several species of birds and mammals characteristically feed almost entirely on herbaceous vegetation, at least at certain times of the year (e.g. the Canada goose, the American wigeon, the fulvous whistling duck, and various voles and rabbits). Therefore, to weight the analysis to address only certain food habits is not justifiable and could lead to erroneous conclusions.

Cheminova Comment: The use of a point estimate of the EEC for calculation of avian reproduction RQs is inappropriate; the EEC should be based on some average over time that accounts for degradation and foliar dissipation of the compound.

EPA Response: Cheminova's comment does not appear to have factored in some important points. First, as Sturkie (1986) points out, physical and biochemical events preceding and contemporary with significant reproductive events in birds need to be considered. This information suggests that there are a number of processes important to the onset of folicular formation, ovulation, eggshell formation, and spermatogenesis that could be open to interference by xenobiotics, and that the possibility for short-term disruptions of these processes could have profound implications for the overall reproduction process. Indeed, the development of the ovarian follicle, ovulation, and egg laying may only span two or fewer weeks and all subsequent effects observed in embryos and hatchlings may be the result of exposure during this short phase, or during any point in the overall life cycle critical to reproduction. Certainly, the work of Bennet and Bennet (1990) with methyl parathion suggests that reproduction impairment can occur after exposure periods much shorter (only eight days) than the currently employed testing guideline but at comparable dietary concentrations.

Secondly, for most pesticides, the toxicological data are not sufficient to characterize the duration of exposure required to induce reproductive impairment. The current reproduction tests used to satisfy OPP data requirements do not allow for discrimination between effects expressed from short-term exposure and effects requiring long-term exposure. The tests do not allow for the identification of critical exposure timing.

If EPA's current screen suggests that exposure may pose reproduction risk, further discussion and characterization of the potential risk is included. This discussion may consider information on the use and environmental fate of the chemical, and environmental conditions that affect exposure levels and exposure duration. This discussion may also characterize the reproductive and sublethal risk in context of the range of possible exposure levels on food items and the distribution of possible exposure levels across food items, under various conditions, and over time.

In general, the short-term EEC will be used for the initial screen unless scientifically sound toxicity data are available that clearly identify the length of time required to cause reproduction effects and identify the critical period in the life cycle for the species at risk.

Cheminova Comment: EPA's attempts to estimate LC50 values for mammals based on LD50 data introduce an inherently large amount of uncertainty into the risk evaluation that is not acknowledged by EPA. Often such estimated LC50s are substantially lower than actual LC50s determined in testing (Hall and Fischer, 1997). EPA should acknowledge the large amount of uncertainty associated with its estimated LC50 values.

EPA Response: EPA acknowledges the uncertainty in using a single oral dose acute toxicity endpoint (an LD_{50}). However, EPA does not have acute dietary toxicity data for methyl parathion upon which to base the risk assessment.

EPA has investigated the cited source of the 96-hour LC50 value for laboratory rats presented in the EPA risk assessment as well as the two laboratory rat LC50 values cited in Cheminova's alternative risk assessment. In both cases, the citation is McCann et al. (1981; MRID43961101). This paper contains neither acute single oral dose nor acute dietary toxicity data for methyl parathion. Because no acute dietary toxicity data are available in either the EPA or Cheminova risk assessments, EPA is compelled to use the available LD_{50} data. EPA will however acknowledge in revised versions of the risk assessment the uncertainty associated with such an approach.

Cheminova Comment: According to two of Cheminova's citations (Brewer, et al., 1997; Fischer, et al., 1997), residues on insects will be one or two orders of magnitude less than would be estimated by grouping insects with plant feed items. In addition, insects should not be used to consider longer term risk, as the treated insects will not be available as a feed item longer-term.

EPA Response: In 1986, EPA established the Standard Evaluation Procedure for Ecological Risk Assessment (EPA-540/9-85-001). This procedure used the Hoerger and Kenaga (1972) data for residues on forage as an estimate for small insects. This decision is supported by the position of Kenaga (1973), which states:

"Initial residues on insects are probably in the same order as those on plants of similar surface area to mass ratios... Most of the factors which affect the decline of residues on plant surfaces are also operative for insect surfaces and so inert residues may be estimated on the basis of insect species having a surface to mass ratio similar to those of equivalent plant type...."

Kenaga (1973) goes on to develop categories of residues with groupings of residue equivalency that include dense foliage and insects together as well as seeds, fruit, and large insects together. Kenaga's (1973) findings have been applied to the data summarized by Fletcher et al. (1994), yielding the preliminary risk assessment assumptions of residue equivalence between broadleaf/forage plants and small insects as well as between fruits, pods, seeds, and large insects.

EPA is open to consideration of any technically valid and statistically robust studies of residues on avian food items. The studies cited by the registrant were not specific to methyl parathion. Therefore, they will be considered in the future with the full body of available exposure data as EPA refines its exposure assessment processes.

Although there is additional uncertainty concerning chronic exposure to contaminated insects as feed, it is not inappropriate to estimate this potential risk for methyl parathion. First, most labeled uses of methyl parathion provide for multiple applications, which can lead to de facto opportunity for chronic exposure. Second, as detailed in the preliminary risk assessment and in this comment response, methyl parathion has caused chronic effects in birds from short-term exposure.

Cheminova Comment: When toxicity endpoints are compared to field trial residue data, there is less apparent risk. The "actual measured residues on herbivorous and seed feed items following maximum application scenarios are significantly lower than EPA's standard estimated residues for these types of seeds."

EPA Response: It is not appropriate to compare the referenced field trial residue data with residues estimated using the Kenaga and Fletcher nomographs. For instance, the cottonseed residues cited by Cheminova, which had a maximum concentration of 4.51 ppm, were for cottonseed that had been ginned and delinted. Animals in the field would be exposed to methyl parathion in the "gin trash" (cotton fibers, stem parts, etc) which is removed by the ginning process. No residue data were provided by Cheminova for gin trash, but pesticide residues are invariably higher in gin trash than in cottonseed. Current pesticide tolerances in 40 CFR 180 for cotton gin trash, which reflect residues after waiting for a number of days equivalent to the pre-harvest interval (PHI), range from 25 to 100 ppm. The estimated residue concentration of 60 ppm for cottonseed, therefore, is not unreasonable, and perhaps is not even conservative.

The field trial data for wheat grain, forage, hay and straw cannot be directly compared to the estimates in the preliminary risk assessment, either, because they do not represent "maximum application scenarios" as Cheminova claims. The residues cited were derived from field trials in which four weekly applications were made at 1.25 lb ai/acre (the maximum label rate), one application at 0.75 lb ai/acre, and one at 0.25 lb ai/acre (not 0.5 lb ai/acre as stated in the comment). The day 0 sample was taken after the final application of 0.25 lb ai/acre. Therefore, the residues cited by Cheminova reflect this low rate application and whatever residues remained from the previous applications. In addition, the hay and straw samples are not taken the day of the final application, but some number of days after. The hay and straw are not sampled until they reach the level of dryness necessary to be used as livestock feed.

There appears to be an inconsistency between the residue value suggested by the registrant for wheat grain and the residue data submitted to the Agency in support of tolerances. The comment cites day 0 residues ranging from 0.07 to 4.39 ppm. However, after reviewing the same data submitted by Cheminova for support of tolerances, the Agency indicated that it will base the tolerance for wheat grain on a concentration of 5.09 ppm measured 15 days after application (which reflects the PHI). The day 0 concentration would have been considerably higher.

Cheminova Comment: The actual measured values in the field trial data "show no indication of residue build-up with multiple applications".

EPA Response: The field trial data provides no basis for this claim. The day 0 samples in these trials were taken only after the final application had been made. Since samples were not taken after each application, it is not possible to determine whether residues from previous applications persisted and were reflected in the day 0 concentration.

Cheminova Comment: "EPA's text concerning exposure to methyl paraoxon in addition to parent methyl parathion is misleading because this text suggests that exposure to methyl parathion should be added to the initial estimated values for methyl parathion . . . The conversion of methyl parathion to methyl paraoxon is not 100%, as implied by EPA's text."

EPA Response: The EPA preliminary risk assessment does not predict nor imply a 100% conversion of methyl parathion to methyl paraoxon. In fact, the environmental fate section specifically describes the maximum amount of methyl paraoxon that was observed in laboratory studies as a percentage of applied methyl parathion. Rather, the chapter states that methyl paraoxon "may form on plant foliage after the parent degrades." Methyl paraoxon from previous applications could be present when the next application of methyl parathion is made. No attempt was made to predict the mass of methyl paraoxon that might be detected in the field. To the extent that the degradate might be encountered by animals in the field, the exposure to methyl paraoxon in addition to methyl parathion will result in additional risk.

Cheminova Comment: "Cheminova is concerned that there is a significant amount of unsubstantiated speculation in this preliminary risk assessment. Key examples include EPA's claims concerning indirect effects on organisms . . . and EPA's hypotheses about tank mixing of different pesticide products and/or sequential applications of different products to a crop.".

EPA Response: EPA has only noted potential indirect effects on organisms when studies were available which indicated they were possible. For instance, studies cited in the risk assessment indicate that a suite of effects occur in birds with short exposure to methyl parathion. These include direct mortality, as well as sublethal effects such as:

- reproduction effects,
- changes in maternal care and viability of young birds,
- anorexia,
- increased susceptibility to predation, and
- greater sensitivity to environmental stress, such as cold.

Indirect mortality to fish from an algae bloom after elimination of aquatic invertebrates was reported as having been observed in an open literature study, and was included as an illustration of potential effects. Similarly, another study attributed growth reduction in trout to a reduction in the invertebrate food supply. The use of scientific papers does not constitute "unsubstantiated speculation"; the information was included as supplemental information to the risk assessment.

In addition, EPA at this time does not believe it is unsubstantiated speculation to expect increased risk when methyl parathion is used in combination with other acetylcholinesterase inhibitors. The extent to which risk is increased will be considered in the cumulative assessment for the organophosphates. In addition, EPA cites studies in which increased toxicity was observed. EPA stands by its original assessment.

Cheminova Comment: "EPA's use of the Breeding Bird Survey to support claims that methyl parathion is responsible for declines for some of the species list . . . represents a misuse of the Breeding Bird Survey Data." "EPA's risk quotient (RQ) procedure is focused on effects at the individual level of biological organization, not the population level; EPA's claims of population level impacts are unsupported by the available data, and because EPA has not performed a rigorous population level analysis, it should refrain from such speculations."

EPA Response: EPA does not claim the declines were caused by methyl parathion. The chapter states that "While these data *do not establish causality for population declines (a variety of factors are likely to contribute to population declines)* (emphasis added), they do suggest that many bird species at a state-wide level of resolution could be sensitive to additional acute or reproductive effects from exposure to methyl parathion.

EPA agrees that the risk index method is a model most appropriate for predicting effects to individual birds. However, EPA currently does not have established tools to address pesticide effects to populations and higher levels of biological organization. Available incident data suggest that individual effects (eg mortality) are being observed in populations of birds associated with agroenvironments treated with methyl parathion.

Cheminova Comment: The registrant disputes the contention that methyl parathion will cause reproductive effects at acute levels. They claim that the study cited by EPA includes a test concentration of 400 ppm, "which is almost two orders of magnitude higher than the concentration EPA proposed for evaluating longer-term effects of methyl parathion; this concentration is at or above the reported acute dietary LC50 for mallards, and nearly 20 times the value EPA claims represents the avian LC50. They also suggest that EPA inappropriately considered the effects reported in Bennet et al. (1990) as being the result of means testing rather than the regression analysis actually used by the study authors.

EPA Response: EPA has reevaluated the study cited as Bennet et al. (1990) in the preliminary risk assessment and agrees that the results presented in the cited study regarding reproduction effects in birds are based on regression analysis and not the typical means testing used by EPA to establish no observed effects levels (NOEL) or lowest observed effect levels (LOELs). However, this study is not the only reported short-term methyl parathion reproduction study conducted in 1990. Bennet and Bennet (1990) report on a short-term reproduction study involving an 8 day *ad libitum* exposure of egg-laying bobwhite quail (8 per control and 6 per each treatment) to dietary concentrations of methyl parathion (0, 14, 20, 28, and 40 ppm). The results of this study are compatible with means testing and indicate a significant ($p < 0.05$) reduction in egg production relative to controls at the 14 ppm treatment level. The results of Bennet and Bennet (1990) suggest that reproductive effects can indeed occur as a result of short-term exposure to dietary concentrations of methyl parathion close to the LOEC (15.5 ppm) established for the current long-term exposure avian reproduction study (MRID 41179302). Consequently, the registrant's reference to the very high dietary concentrations of methyl parathion (400 ppm) reported to be

associated with short-term exposure reproduction effects in mallards from Bennet and Williams (1991) as a refutation of the reasonable possibility for short-term exposure effects on reproduction appears to be moot.

World Wildlife Fund Comment: The World Wildlife Fund (WWF), supported by other non-governmental organizations such as the Consumers Union and Natural Resources Defense Fund, comments that "there is substantial evidence that in addition to the developmental effects that have been described in the preliminary risk assessment, methyl parathion exhibits clear signs of endocrine disruption, both in vitro and in vivo." The WWF suggests that methyl parathion should be among the chemicals tested when EPA's Endocrine Disruptor Screening and Testing Advisory Committee (EDSTAC) agrees to test for endocrine disrupting properties.

EPA Response: The body of data provided by WWF, in conjunction with that found in the preliminary risk assessment, provides evidence of possible endocrine disruption by methyl parathion. Methyl parathion has been observed in the open literature to display metabolic effects which hinder successful reproduction and/or sexual development in birds, mammals, fish. The observations included the following:

1. Damage to oocytes in fish (Rastogi and Kulrestha, 1990)
2. Interference with spermatogenesis in rats (Zlateva and Moleva, 1976)
3. Decreased testes weight and function in birds (Maitra and Sarkar, 1996)
4. Serum and pituitary gland gonadotropin hormone decreases in fish (Ghosh, et al., 1990)
5. Interference with glucose metabolism in rats, snails, prawns, and birds. (Lukaszewicz-Hussain, et al., 1985, Reddy and Rao, 1991 and Rambabu and Rao, 1994.)
6. Elicitation of strong estrogenic response in liver hepatocyte cells, possibly due to a metabolite (Petit, F., et al., 1997)
7. Disruption of eggshell formation in birds (Bennett and Bennett, 1990).

The amendments to the FQPA and the Safe Drinking Water Act (SDWA) mandate or support the development of a screening program that will determine whether pesticides and certain drinking water sources contaminants "may have an effect in humans that is similar to an effect produced by a naturally-occurring estrogen, or other such endocrine effect as the Administrator may designate." Very early in its deliberations, EDSTAC determined that there was both a strong scientific basis and feasibility, considering time and resource constraints, to expand the scope of the screening program to include the androgen- and thyroid hormone systems, and to include evaluations of the potential impact on wildlife as well as on human health. EPA agrees an is developing a screening program which incorporates these modifications.

Based on the adverse results observed in the above data, EPA will ensure that when the Endocrine Disruptor Screening Program (EDSP) is implemented, methyl parathion will be subjected to more definitive testing.

Cheminova Comment: "EPA's discussion of the sublethal effects of methyl parathion on birds is misleading because EPA presents these as effects that only occur following exposure to methyl parathion."

EPA Response: EPA's assessment was based on literature which considered the reported effects on birds following methyl parathion exposure and compared these with controls. EPA agrees that sublethal effects may be observed for other compounds, but this observation is not relevant to methyl parathion. EPA stands by its assessment of sublethal effects caused by exposure to methyl parathion.

Cheminova Comment: The registrant noted that application efficiency for airblast and aerial application to orchard crops was not factored in the avian and mammalian risk assessment. They stated that the application efficiency for air-blast and aerial applications on orchard crops is 50%. Because application efficiency was not considered in the assessment, the registrant believes the terrestrial exposure assessment is highly conservative.

EPA Response: As noted in an earlier comment, EPA uses empirical data for determining estimated environmental concentrations in terrestrial environments. Implicit in the empirical data is application efficiency as based on research data using foliar concentrations on different crop groupings from normalized pesticide application rates. Because the exposure assessment is based on pesticide concentration, there is no attempt to construct a mass balance in the terrestrial exposure assessment. Additionally, the terrestrial exposure assessment assumes overspray from the target crop is likely and hence will result in concentrations predicted through the empirical data. Moreover, virtually all droplets impinge on surfaces that could result in bird exposure, regardless of whether it is directly on the crop or not. Finally, there were no data submitted which support the contention of a 50% application efficiency for aerial and airblast applications.

Cheminova Comment: Cheminova requests that EPA provide references supporting its estimated daily feed consumption values as a percentage of body weight, particularly because EPA's estimates of daily feed consumption are much higher that values typically found in the published literature, including values referenced in EPA's 1986 Ecological Risk Assessment Standard Evaluation Procedure and EPA's 1993 *Wildlife Exposure Factors Handbook*.

EPA Response: EPA has used the allometric equation from Nagy (1987) for dry weight ingestion rates as follows:

$$\text{ingestion rate (dry weight, g/day)} = 0.621\ (\text{body weight g})^{0.564}$$

Contrary to Cheminova assertions, this equation is presented in the USEPA (1993) *Wildlife Exposure Factors Handbook* (equation 3-8, page 3-6). Because this equation yields a body-weight dependent estimate of ingestion rate in terms of dry-weight for the food item, an adjustment must be made to account for the fresh-weight food item encountered by wildlife in the field. This is accomplished through the following equation:

$$\text{ingestion rate (wet-weight, g/day)} = \frac{0.621\ (\text{body weight g})^{0.564}}{1 - \text{fraction water content of food item}}$$

EPA has assumed the following fraction water contents for the various diets:

herbivore diet: 80 % water
insectivore diet: 80 % water
granivore diet: 10 % water

These assumptions of water content are supported by data presented in the USEPA (1993) *Wildlife Exposure Factors Handbook*

Dietary Items	Water Content
forage:	young grasses 70 % - 88 % (Table 4-2, USEPA 1993)
	dicot leaves 85 % (Table 4-2, USEPA 1993)
terrestrial invertebrates:	earthworms 84 % (Table 4-1, USEPA 1993)
grasshoppers/crickets	69 % (Table 4-1, USEPA 1993)
beetles	61% (Table 4-1, USEPA 1993)
seeds:	9.3 % (Table 4-2, USEPA 1993)

Using the Nagy (1997) allometric equation and a herbivore dietary water content of 80% the following fresh-weight dietary mass and percentages of body weight are calculated

Herbivore Body Weight	Daily Ingestion	% Body Weight Ingested
15 g	14.3	95.3
35 g	23.1	65.9
1000 g	152.8	15.3

Using the Nagy (1997) allometric equation and a herbivore dietary water content of 10% the following fresh-weight dietary mass and percentages of body weight are calculated

Herbivore Body Weight	Daily Ingestion	% Body Weight Ingested
15 g	3.18	21.2
35 g	5.13	14.6
1000 g	34.0	3.4

These food ingestion rate and percent of body weight values are consistent with the values used in the methyl parathion preliminary risk assessment.

Cheminova Comment: Terrestrial acute exposure values used by EPA are based on overly conservative maximum Kenaga/Fletcher values.

EPA Response: EPA believes that a discussion of the validity of the use of Kenaga nomograph values, as modified by Fletcher et al. must recognize that the values are based on a robust set of actual field residue data. Hoerger and Kenaga (1972) state that the upper limit values from the nomograph represent the 95th percentile of residue values from actual field measurements. The Fletcher et al. (1994) modifications to the Kenaga nomograph are also based on measured field

residues from 249 published research papers, including information on 118 species of plants, 121 pesticides, and 17 chemical classes. These modifications represent the 95th percentile of the expanded data set.

Because pesticide regulatory decisions involve potentially widespread uses of pesticides, EPA believes that the use of upper limit values is necessary to account for the potential variability and uncertainty associated with application to a wide variety of use sites under a variety of environmental conditions. However, EPA will consider chemical- and use-specific residue data, provided the data set is sufficiently robust to account for intra- and inter-site variability as well as account for temporally variable environmental conditions. Unless such data are submitted, EPA will continue to use the Kenaga nomograph values, as modified by Fletcher et al.

Cheminova Comment: Cheminova disagrees with EPA's postulation that the bioconcentration of methyl parathion in carnivorous/piscivorous feed items such as fish or tadpoles represents a significant exposure pathway of birds, mammals or other fish because methyl parathion has a low bioaccumulation potential and is rapidly metabolized and excreted by fish." Additionally, the study cited in the preliminary risk assessment dealt with concentrations in the 1 to 5 mg/l range, which is greater than the calculated EECs.

EPA Response: EPA agrees that the EECs presented in the preliminary risk assessment were less than the 1 to 5 mg/L range reported in the study. This study had been cited because waterfowl in the prairie-pothole region can be found in water bodies shallower than that simulated by PRZM and EXAMS, and therefore could potentially be exposed to concentrations higher than those predicted by the models. However, after considering the concentrations in the study along with the additional uncertainty from the extrapolation of effects from the organophosphate chemicals in the cited study to methyl parathion, EPA will remove the bioconcentration section from the preliminary risk assessment

The suite of sublethal effects to birds that can be attributed to methyl parathion is still extensive, though, and the basic conclusions of EPA's avian risk assessment for methyl parathion are not altered greatly by these changes.

Cheminova Comment: "Cheminova sees little relevance for the cold stress factor cited by EPA as an additional element that could increase the avian risk assessment for methyl parathion because methyl parathion applications to crops are not made under cold stress conditions. Rather, methyl parathion is applied primarily in warmer climatic conditions, when target insect activity is high."

EPA Response: The two studies cited under the section "Increased Toxicity from Environmental Stress" are included as evidence that laboratory toxicity tests may underpredict methyl parathion toxicity in the field. The following language will be added to this section: "Methyl parathion is unlikely to be applied when the temperature outdoors is below freezing. However, these studies suggest that environmental stresses may reduce the amount of methyl parathion needed to cause intoxication or mortality below concentrations indicated by laboratory studies."

Comments on the Risk of Methyl Parathion to Non-Target Insects

Cheminova Comment: Penncap-M has not been a serious problem for bees since 1992.

Several Comments: Penncap-M continues to kill bees, and should be removed from the market.

EPA Response: There is evidence in the incident table included in the preliminary risk assessment that Penncap–M has continued to cause bee kills since 1992. In addition, several beekeepers provided comments in which they described damage to their colonies associated with methyl parathion.

However, although the use of encapsulated methyl parathion on field crops will continue to pose a risk to bees, **the voluntary cancellation of tree fruit uses of methyl parathion will result in a significant reduction in the overall risk to bees.** Of the 22 incidents listed in the table included with the preliminary risk assessment, 19 were associated with the use of Penncap-M on orchard crops.

Comment: "The American Beekeeping Federation Survey does not provide a statistically relevant portrayal of the situation." The information provided in the preliminary risk assessment "is missing some key information that is necessary to fully evaluate EPA's claims concerning bee incidents. Most significantly, this table fails to provide any information concerning the number of beekeepers that the survey was sent to, or even data about the number of beekeepers in each of the states listed." In addition, the survey listed bee colonies damaged in states where the no colonies are reported to be in operation.

EPA Response: The extreme toxicity of methyl parathion to bees, when considered with the extensive database of bee kills caused by Penncap-M, is sufficient evidence to conclude that methyl parathion poses high acute risk to bees. The survey was included in the preliminary risk assessment as additional evidence that beekeepers continue to report problems with Penncap-M, even after the well-known problems reported in 1992.

EPA reported in the preliminary risk assessment that 60 beekeepers, operating 127,950 colonies in 22 states, reported significant bee losses from pesticides. EPA will add the information that 26 beekeepers, operating 16,439 colonies, did not believe that pesticide losses were significant to their operations. EPA does not have information on the total number of beekeepers in each state. EPA will add a footnote that was inadvertently omitted in the preliminary risk assessment:

> * Migratory beekeepers reported losses in some states where no resident beekeepers responded.

EPA agrees that the results of the survey are not statistically robust. As suggested in other comments received by EPA, many bee kills may go unrecognized or unreported. The true extent of methyl parathion-related bee kills cannot be determined by the results of the survey, nor by the number of bee kills included in the incident database.

Cheminova Comment: Not all bee incidents reported in the preliminary risk assessment actually show effects. EPA also assumes that incidents that include multiple chemicals were caused by methyl parathion.

EPA Response: The evidence **is very strong for many of the bee kills reported.** Most did involve detections of methyl parathion in dead bees, pollen, honey, and/or wax at the hive. As mentioned above, the most common crop use reported was on tree fruits, which have been voluntarily cancelled. However, the preliminary risk assessment also details incidents involving corn or alfalfa, which are also frequented by bees. Methyl parathion detections on local orchard vegetation were also common, along with occasional detections of other insecticides. In those cases where multiple pesticides were detected, the methyl parathion residues found in the bees were sufficient to kill them, based on the toxicity data referenced in the preliminary risk assessment.

Many of the incidents in the table contain the entry "Not reported" in the "Effect/#" field. This is not an indication that there was no effect, but an indication that the number of dead bees was not reported. The majority of incidents that include the "Not reported" entry report concentrations of methyl parathion in dead bees.

Several Comments: Label language should indicate the duration of toxicity of methyl parathion to bees. Also, it should be made clear that "bloom needs to be controlled before pesticide applications are made". Blooming weeds among the target crop, as well as the blooms of flowering crops, are potential sites for honeybee poisoning with methyl parathion.

EPA Response: The Agency is currently working with Association of American Pesticide Control Officials (AAPCO) and SFIREG (in conjunction with AAPCO) to develop label language describing hazard to bees. In the near future, the Agency will present this proposed language to stakeholders for their input.

Cheminova Comment: "The statement on the EECs calculated for bees and beneficial insects is inaccurate. How does EPA define the level of concern? On page 39, EPA indicates that it does not currently perform risk assessments for nontarget insects. In contrast,... EPA's claims for EECs for bees infers that EPA has performed some sort of quantitative assessment for non-target insects. The *1998-1999 Pennsylvania Tree Fruit Production Guide...* clearly demonstrates that Mcap is slightly toxic to beneficial insects in comparison to the majority of the alternatives."

EPA Response: After consultation with BEAD, EPA agrees that it was incorrect to extrapolate honey bee toxicity data to other beneficial insects in general. The discussion of risk to beneficials other than honey bees will be revised. The preliminary risk assessment cited one study from the open literature (Brown, et al., 1978) which indicated that predators of a cereal aphid were highly susceptible to methyl parathion exposure.

The preliminary risk assessment should not have stated that EECs exceeded LOCs for honey bees. The risk to honeybees is known with greater certainty due to actual foliar residue studies

submitted to and validated by the Agency. Atkins and Kellum (1980) reported that residues of methyl parathion on alfalfa foliage were highly toxic to honeybees at application rates ranging from 0.03125 to 0.5 lb ai/acre. At the higher rates (0.25 and 0.5 lb ai/acre), the toxicity persisted from 4 to 6 days. This will be clarified in the revised risk assessment.

The language stating that "EPA does not currently perform risk assessments for nontarget insects" was not sufficiently precise. The section of the preliminary risk assessment cited by Cheminova describes the risk index method used for assessment of risk to birds, mammals and aquatic animals, but not for nontarget insects. Since EPA clearly does perform a risk assessment for honeybees, the revised risk assessment will be amended to state that EPA does not perform a "similar" (risk index method) risk assessment for nontarget insects.

Data Gaps

Comment: EPA claims that the estuarine/marine invertebrate chronic toxicity study is required in the transmittal memo, but says it is fulfilled in the chapter itself.

EPA Response: As Cheminova notes, the estuarine/marine invertebrate chronic toxicity study requirement is fulfilled. The estuarine/marine fish chronic toxicity study is still outstanding.

Comment: EPA requires nontarget terrestrial plant testing on page 68, but says it is not required on page 38.

EPA Response: Data from Youngman, et al. (1989) presents a compelling case that methyl parathion can cause growth reduction in terrestrial plants. The nontarget terrestrial plant studies are required as stated in the preliminary risk assessment. The language on page 38 will be revised to reflect this requirement.

Comment: Cheminova challenges the need for generating additional data on the affects of methyl parathion on algae.

EPA Response: EPA will reserve the aquatic plant testing requirement, pending the results of the terrestrial plant studies required as described above.

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